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Case report

Cerebellar infarction presenting as inner ear decompression sickness following scuba diving: A case report

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ABSTRACT

Introduction: Inner ear decompression sickness following scuba diving is not uncommon and the characteristic features of this disorder are acute peripheral vestibular syndrome, sometimes associated with cochlear signs, requiring urgent hyperbaric oxygen therapy. Cerebellar infarction can also mimic isolated peripheral vestibulopathy.
Case report: The authors report the case of a 47-year-old man in good general health admitted with acute left vestibular dysfunction suggestive of inner ear decompression sickness 6 hours after scuba diving. Normal videonystagmography and delayed onset of occipital headache finally led to brain MRI that confirmed the presence of recent ischaemic infarction in the territory of the medial branch of the posterior inferior cerebellar artery. Complementary investigations revealed the presence of a patent foramen ovale with atrial septal aneurysm. No underlying atherosclerotic disease or clotting abnormalities were observed.
Discussion/Conclusion: Cerebellar infarction can present clinically with features of inner ear decompression sickness following scuba diving. An underlying air embolism mechanism cannot be excluded, particularly in patients with a large right-to-left circulatory shunt and no other cardiovascular risk factors.

1. Introduction

Scuba diving is associated with a risk of decompression sickness due to excessive formation of gas bubbles (mainly nitrogen) in the body on return to the surface. This fairly uncommon acute illness (1 to 3 cases per 10,000 dives) mainly affects the central nervous system [1]. In 25–30% of cases, the inner ear is involved by *in situ* release of bubbles into endolymph or perilymph or air embolism in the labyrinthine artery [2,3]. The diver generally experiences varying degrees of rotational vertigo sometimes associated with cochlear signs during the hour following return to the surface. The diagnosis is based on the presence of peripheral vestibular syndrome confirmed by videonystagmography (VNG). In 80% of cases, the patient presents a right-to-left circulatory shunt [3]. Cases of neurological decompression sickness with cerebellar lesions have been described, but they are exceptional and are associated with clinical signs of the central lesion [4,5].

Cerebellar infarctions represent only 3% of all ischaemic strokes [6]. In 11% of cases, they present in the form of isolated acute vestibular syndrome with ischaemic lesions predominantly demonstrated in the territory of the medial branch of the posterior inferior cerebellar artery (PICA) [7]. This particular clinical form resembling the features of vestibular neuritis can sometimes lead to an incorrect diagnosis, as the central or peripheral origin of the lesion cannot be distinguished on neurological examination [8].

We report the case of a scuba diver admitted with clinical features suggestive of inner ear decompression sickness, but in whom the work-up finally demonstrated cerebellar infarction due to thrombosis of the medial branch of the PICA. We discuss the possibility that this cerebellar infarction may have been initiated by air embolism generated following diving.

2. Case report

Mr R., 47 years old, was referred to our department with suspicion of inner ear decompression sickness after scuba air diving (17 min at a depth of 44 metres sea water with a 4-minute decompression stop at 3 metres) conducted under optimal conditions with adequate decompression. The patient was a non-smoking military sportsman with no particular history.

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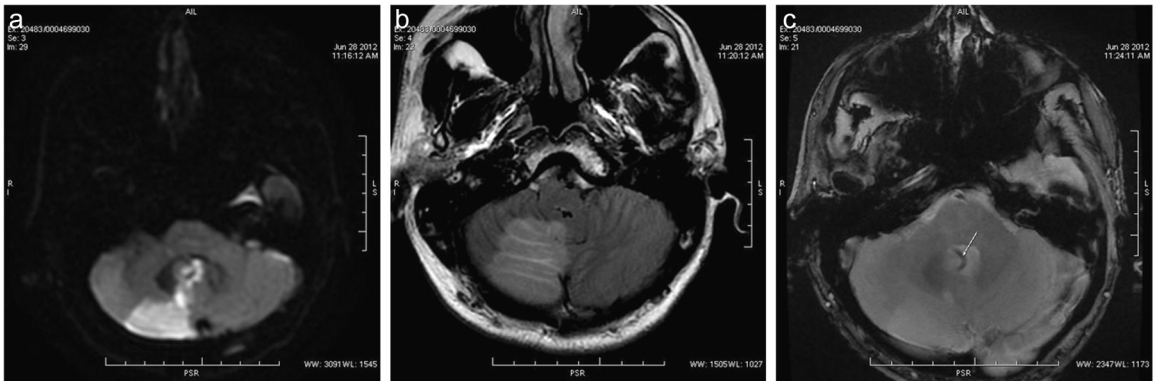

Fig. 1. Clearly demarcated zone of high-intensity signal in the medial postero-inferior aspect of the right cerebellar hemisphere on diffusion-weighted (a) and FLAIR sequences (b) consistent with recent infarction in the territory of the right posterior inferior cerebellar artery. (c) Arcuate low-intensity signal on the T2-weighted sequence corresponding to thrombus in the right posterior inferior cerebellar artery (white arrow).

Six hours after leaving the water, the patient suddenly experienced vertigo accompanied by vomiting, requiring rapid transfer to our department on high-flow oxygen.

On admission, the prostrate patient presented intractable vomiting and intense rotational vertigo and was unable to remain standing. Clinical ENT examination demonstrated grade II spontaneous right horizontal nystagmus visible with and without videonystagmoscopy glasses, associated with falling to the left while sitting. No multidirectional (gaze-evoked) nystagmus or oculomotor disorders were observed. Otoscopy excluded the presence of barotrauma to the middle ear. Neurological examination was normal with no other cranial nerve lesions or disorders of coordination.

A diagnosis of left vestibular decompression sickness was proposed in this context of apparently peripheral vestibular syndrome. The patient was treated by hyperbaric oxygen therapy associated with intravenous drug treatment (metoclopramide 10 mg, acetylleucine 500 mg and methylprednisolone 80 mg). Only limited clinical improvement was observed after leaving the hyperbaric chamber, as is generally the case with this type of accident [3]. The patient was hospitalised for subsequent care (hyperbaric oxygen therapy sessions, vestibular rehabilitation) and further assessment. During the night, he reported occipital headache and, on the following day, marked left axial deviation (70°) was observed on Fukuda stepping and Babinski-Weill postural tests.

Systematic investigation for right-to-left shunt by transcranial Doppler revealed the passage of a large quantity of contrast bubbles during spontaneous breathing, suggesting a possible underlying patent foramen ovale. The unexpected normality of complementary examinations (VNG with eye tracking study and bithermal caloric tests, audiometry) performed after 72 hours despite persistence of spontaneous right nystagmus constituted an indication for brain MRI looking for a central cause. MRI confirmed the presence of recent cerebellar infarction in the territory of the medial branch of the right PICA with an associated thrombus image (Fig. 1). Computed tomography of supra-aortic vessels excluded vertebral artery dissection. On the 5th day, the patient presented a favourable clinical course and only presented mild ataxia on walking. Cardiological investigations performed after discharge from hospital, looking for emboligenic heart disease (transthoracic echocardiography, Holter ECG and transoesophageal echocardiography) confirmed the presence of a large patent foramen ovale associated with atrial septal aneurysm (ASA). Comprehensive assessment of thrombophilia failed to demonstrate any particular laboratory thromboembolic risk factors.

At 3 months, the patient reported only occasional feelings of instability on effort. He had returned to work with platelet aggregation inhibitor and permanent incapacity for scuba diving.

3. Discussion

Despite the relatively delayed onset of clinical signs, this patient presented acute vestibular disorders following scuba diving, for which the most likely diagnosis was inner ear decompression sickness requiring emergency recompression therapy. The possibility of a cerebellar lesion was only considered and subsequently confirmed by imaging in the absence of VNG abnormalities and the delayed onset of occipital headache. The Halmagyi-Curthoys test performed on admission would have been able to specifically distinguish a peripheral vestibular lesion from cerebellar infarction, but unfortunately it was not performed [7,8]. Cerebellar infarction presenting with features of isolated vestibulopathy is rare and is essentially observed in the case of a focal lesion in the PICA territory [9].

The discovery of a large patent foramen ovale with ASA in this diver with no known cardiovascular risk factors was strictly compatible with the initial diagnosis of inner ear decompression sickness [2,3], but this association is also frequently observed in embolic cerebellar strokes in subjects younger than 40 [10].

Decompression illness is believed to be associated with thrombotic phenomena secondary to the presence of intravascular bubbles. It is generally accepted that mechanical abrasion of bubbles on the vessel wall induces endothelial dysfunction and activation of blood cells responsible for an inflammatory reaction, vasomotor disorders, platelet aggregation and activation of coagulation [11]. As a thrombus was visible in the right PICA, the hypothesis that an arterial thromboembolic event may have been triggered or facilitated by air embolism is a possibility that cannot be excluded.

4. Conclusion

Cerebellar infarction can present with the clinical features of inner ear decompression sickness following scuba diving. The unusual onset of occipital headache and the discordance between clinical findings and the normal VNG should raise the suspicion of a central lesion, requiring emergency brain MRI looking for an ischaemic stroke in the territory of the PICA that may require thrombolytic therapy. An air embolism predisposing factor cannot be excluded, particularly in a patient with a large patent foramen ovale.

Disclosure of interest

The authors declare that they have no conflicts of interest concerning this article.

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